from the beach waters where he regularly surfed, which was near an area of sewage effluent. Non-01 *Vibrio cholerae* was isolated not only from the sea water but eventually from two nearby freshwater creeks as well, about 5 and 25 miles from the site of original contamination.

Discussion

De Gerome and Smith[1] reported the first case of non-01 vibrio infection contracted in this country in a patient who had visited New Orleans and in whom a prolonged diarrheal illness developed. Hughes and associates[2] described 26 patients seen between January 1972 and March 1975 and felt there had been a dramatic increase in the number of non-01 *V cholerae* isolates referred to the Center for Disease Control for identification. Thirteen (50%) organisms were isolated from stool specimens of patients having acute diarrheal illness and most of the patients had a history of recent shellfish or raw seafood ingestion or foreign travel. In 1981 Morris and co-workers reported 14 more sporadic cases of non-01 *V cholerae* gastroenteritis who had diarrhea, abdominal cramps and fever; the patients with domestically acquired cases had a recent history of eating raw oysters, whereas the other patients had traveled outside the United States.[3] Non-01 *V cholerae* gastroenteritis is increasingly being recognized as a clinical entity and can produce an acute diarrheal illness identical to classic *V cholerae or Vibrio parahaemolyticus* gastroenteritis. *V parahaemolyticus* gastroenteritis also begins abruptly with explosive watery diarrhea and abdominal cramps but subsides spontaneously within 24 to 48 hours. *V parahaemolyticus* does not produce a cholera-like enterotoxin whereas non-01 *V cholerae* can produce a toxin virtually identical to the cholera enterotoxin.[4]

Most of the domestically acquired cases have been contracted in the Texas-Louisiana gulf coast region after raw seafood ingestion.[3] The actual incidence of non-01 *V cholerae* gastroenteritis may be even greater than previously recognized because only patients with severe diarrhea and dehydration seek medical help.[5] Systemic non-01 *V cholerae* infections have been described in immunocompromised and debilitated patients after occupational or recreational exposure to salt water without classic gastroenteritis.[2] Surprisingly, the reported incidence of vibrio infections in California is low despite the significant numbers of people traveling in and out of this state from other countries. Our one case represents an unusual exposure to the pathogenic organism related to offshore surfing near an area of sewage effluent. In none of the other surfers using this area did any symptoms whatsoever develop and we have not seen any additional cases of similar infection in our county to date.

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Carotid Artery Bruits and Lacunar Strokes

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SEVERAL RECENT SURVEYS have shown a surprisingly high frequency of carotid artery bruits among asymptomatic adults: 2.1% to 3.1% at ages 45 to 54; 4.0% to 4.2%, ages 55 to 64, and 7.0% to 7.1%, ages 65 to 74.[1,2] These bruits may serve more as nonspecific markers for generalized vascular disease than as harbingers of stroke in an area distal to the involved carotid artery.[1-3] Lacunar infarcts appear to be decreasing in frequency, but they are still common, with an autopsy prevalence of 8%.[4] Fisher showed a significant association of atherosclerosis and lacunar disease in a large unselected autopsy series.[5] There appeared to be no correlation between presence or severity of internal carotid atherosclerosis and lacunar disease. However, there is scant mention in the literature of a carotid bruit in association with a lacunar infarct. Herein are reports of cases of two patients with carotid artery bruits who had typical lacunar stroke syndromes ipsilateral to severe internal carotid artery disease.*

Reports of Cases

CASE 1. The patient, a 69-year-old right-handed man, had been known to have moderately severe hypertension for at least 15 years. He had been admitted to hospital in 1971 and 1973 for two episodes of congestive cardiac failure attributed to hypertension. In the late 1970s renal insufficiency developed and he had a serum creatinine level of 2.5 to 3.5 mg per dl. His antihypertensive regimen at the time of his stroke included 40 mg a day of furosemide, 10 mg a day of metolazone, 0.4 mg twice a day of clonidine hydrochloride (β-blockers had caused bronchospasm) and 5 mg twice a day of minoxidil. On this regimen his blood pressure measurements ranged from 130 to 160 mm of mercury systolic and 90 to 100 mm of mercury diastolic.

In addition to hypertension he had a five-year history of type II diabetes mellitus, treated with insulin, and

*Bernard Bodmer, MD, and David Giles, MD, provided technical help in analyzing these cases.

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hypercholesterolemia—his serum cholesterol values ranging from 290 to 330 mg per dl with normal triglycerides. There was an insignificant, remote cigarette-smoking history.

Severe diffuse peripheral vascular disease was present. In 1979 aortography, done because of claudication of both hips and legs, showed severe atherosclerotic disease of the distal aorta and both iliac arteries. In February 1981 he had lower abdominal and back pain and underwent emergency excision of a 5-cm abdominal aortic aneurysm for "impending dissection," from which he recovered without event. In June 1982 sudden weakness and loss of coordination of his right leg developed. He did not seek medical attention, and the weakness resolved over ten days to two weeks. In September 1982 a left carotid bruit was detected. Ultrasonic imaging showed moderate atherosclerosis of both carotid bulbs without identifiable ulceration. Color-coded Doppler imaging showed about 50% to 70% stenosis of the right internal carotid artery and 80% stenosis of the left internal carotid artery at the bifurcation. He refused to consider arteriography and surgical treatment. He had no other neurologic events or amaurosis fugax until January 1983.

In January 1983 he had sudden onset of numbness and clumsiness of his right side. There were no other symptoms. On physical examination the patient was plethoric and had a blood pressure of 180/90 mm of mercury. Cardiorespiratory examination showed no abnormalities except for an S_4 gallop. There were systolic bruits heard over both femoral arteries and no popliteal or pedal pulse could be palpated. There was a systolic bruit over the left carotid artery at the angle of the jaw, but carotid pulses, as well as preauricular and temporal pulses, were full and equal. There was a right hemisensory deficit to all basic modalities with near-perfect midline splitting. His gait was unsteady and he was unable to tandem walk. He could walk on his heels or toes, however, and detailed testing showed no abnormality in muscle strength or tone. Rapid rhythmic alternating movements, finger-to-nose, heel-to-shin and foot-tapping tests were done normally and symmetrically.

There was rapid resolution of the gait disorder and the patient was walking normally by a week. There was slower resolution of the sensory deficit and by four weeks after the ictus there was a barely detectable decrease in sensation to pin and light touch on the right side. A computed tomographic (CT) scan done 14 days after the event showed no abnormalities.

CASE 2.* The patient, a 64-year-old right-handed man, had had known hypertension for at least six years and was being treated with chlorthalidone and clonidine; blood pressure readings ranged from 150 to 180 mm of mercury systolic and 90 to 100 mm of mercury diastolic. He had a 50-pack-year history of smoking. There was no diabetes mellitus or hyperlipidemia. In 1977 a right aortofemoral bypass graft had been done for intermittent claudication. In September 1982 he underwent left iliac artery angioplasty for hip and calf claudication. At that time a left carotid bruit was heard. Ultrasonic imaging showed extensive atherosclerotic disease in the right internal and external carotid arteries, less severe disease in the left internal and external carotid arteries and no ulcerations. He had no history suggestive of transient ischemic attacks, stroke or amaurosis fugax until February 1983.

In February 1983 the patient awakened with weakness on the left side of his body and slurred speech. On physical examination there were bilateral carotid bruits, louder on the right, with normal pulses. Initial neurologic examination showed a left hemiparesis with left central facial weakness, deviation of the uvula to the right, slurred speech, profound weakness of the left arm and leg and a plantar extensor response on the left. He was fully oriented, responded appropriately and was aware of his deficit. He was able to draw a clock face accurately with all numbers, hour and minute hands for several different requested times. Sensation was intact to all basic modalities without extinction. He was able to identify numbers written on either palm and could distinguish among various coins placed in either hand (with help closing left hand and moving coin about). Later in his hospital course there was suggestion of extinction of response to left-sided visual threat and of some left hemineglect. He showed substantial improvement in his weakness after one to two days and by seven to ten days there was only minimal weakness of the upper extremity and he could walk with a cane. He was able to dress, feed and shave himself.

Color-coded Doppler carotid imaging showed an apparent total occlusion of the right internal carotid artery, 50% stenosis of the left internal carotid artery and 70% or greater stenosis of the right and left external carotid arteries. A CT scan done six days after the stroke showed a 1-cm ill-defined lucency in the right basal ganglia (Figure 1).

Discussion

The first case represents a typical example of a pure sensory stroke, which has been excellently reviewed recently.[4,6,7] The remarkable midline split and involvement of face, arm and leg seen in this case are felt to be indicative of ischemia in the thalamus and not the superficial cortical areas or brain stem. CT studies have been unrevealing in nearly all instances. In the few cases studied at autopsy the lesion has been in the ventral posterior nucleus of the thalamus. This area is supplied by the small (100 microns) thalamoperforants arising from the posterior half of the circle of Willis and the stems of the posterior cerebral arteries. The presumed arterial pathology is lipohyalinosis, microatheroma of the penetrating arteries or atherosclerosis of the posterior cerebral artery with involvement of the origin of the penetrator. While microembolization is inferred to be the cause in some cases of lacunar stroke,[8] this is not a likely mechanism for the smaller infarcts causing a pure sensory stroke. Fisher comments in his review of 135 cases of hemisensory symptoms,

*Phillip Levy, MD, gave permission to include this case.

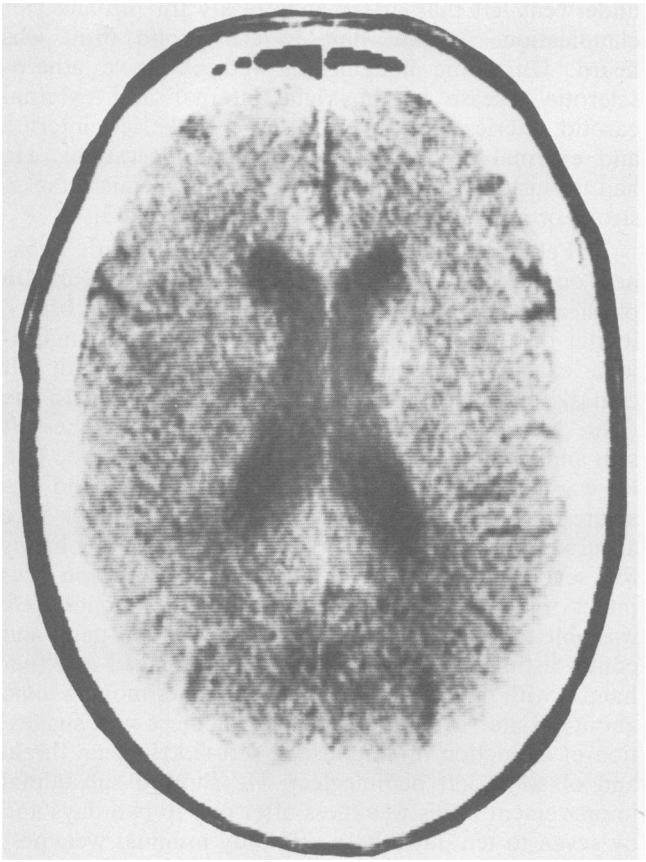

Figure 1.—A computed tomographic scan showing a 1-cm ill-defined lucency in the right basal ganglia.

including 58 patients with pure sensory stroke, that none had carotid disease "by clinical and laboratory assessment short of routine angiography."[7] Angiography was not felt to be indicated clinically but would have helped delineate the extent of carotid artery disease and the relative contribution of the internal carotid and basilar arteries to the posterior cerebral artery. While this patient had a carotid bruit and ultrasonic-Doppler evidence of internal carotid disease on the appropriate side, the infarct was most likely in the thalamus and unrelated to the carotid disease.

The second case is a typical example of pure motor hemiplegia. This syndrome is defined by weakness or paralysis of the face, arm and leg on one side without sensory deficit, hemianopia, aphasia, apraxia or agnosia in the acute phase. Pure motor hemiplegia is caused most often by a capsular infarct, occasionally by a pontine infarct and rarely by a variety of other causes in other areas of the central nervous system.[6] Rascol and co-workers reported on 30 cases of pure motor hemiplegia. No carotid bruits were described, and angiography done in 21 patients did not show carotid artery disease. In 29 of the 30 patients, a lesion able to explain the stroke was found by CT study. They describe three varieties of ischemic capsular lesions: type I, capsulo-putamen-caudate; type II, capsulo-pallidal, and type III, anterior capsulo-caudate. The lesion seen on CT study in case 1 is in the posterior limb of the internal capsule and corona radiata and corresponds best with a type I infarct. This area is supplied by the lateral lenticulostriate branches of the middle cerebral artery. Embolization to this area from internal carotid artery disease is certainly possible, but this seems unlikely in this patient who has apparent occlusion of the internal carotid.

Aleksic and George reported on two cases of pure motor hemiplegia with occlusion of the extracranial internal carotid artery.[10] They assumed a causal relationship in their cases and postulated that a reduction in mean pressures distal to carotid artery stenosis or occlusion might cause thrombosis to occur in the small penetrating arteries supplying the internal capsule. There is no way of proving or disproving this hypothesis, but such a mechanism seems no more likely than fortuitous association. Indeed, Fisher has suggested that internal carotid stenosis might be protective against ipsilateral lacunar strokes by lowering intracranial arterial pressure.[5]

Patients who have an asymptomatic carotid bruit or even documented internal carotid artery stenosis may not be at significantly greater risk for stroke in the territory of that carotid artery than matched controls.[1,2,11] This conclusion is controversial but, if correct, would mitigate against consideration of surgical therapy in an asymptomatic patient. One of the many areas of debate is what symptoms are related to internal carotid disease.[3] The cases reported here represent yet another problem in interpreting symptoms. The lacunar syndromes of pure sensory stroke or pure motor hemiplegia are unlikely to be related to internal carotid artery disease. However, a characteristic of many lacunar strokes—and a feature seen in the cases reported here—is significant improvement in the neurologic deficit over days to weeks. If such patients are not evaluated until some time after the event, then the ability to distinguish a lacunar stroke from cortical ischemia might be quite difficult and the temptation for surgical intervention increased. Awareness of the clinical features of the various lacunar strokes, early detailed neurologic evaluation and later CT scanning should allow distinction between lacunar strokes and symptoms related to internal carotid artery disease.

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